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EXAMINER	
RAE, CHARLESWORTH E	

ART UNIT	PAPER NUMBER
1611	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	**Application No.** 10/685,942	**Applicant(s)** MAN ET AL.	
	Examiner CHARLESWORTH RAE	**Art Unit** 1611	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE _3_ MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on _04 February 2009_.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) _1,5,7 and 8_ is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) _1, 5, AND 7-8_ is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

In view of the Appeal Brief filed on 02/04/09, PROSECUTION IS HEREBY REOPENED. A non-final Office action is set forth below.

To avoid abandonment of the application, appellant must exercise one of the following two options:

(1) file a reply under 37 CFR 1.111 (if this Office action is non-final) or a reply under 37 CFR 1.113 (if this Office action is final); or,

(2) initiate a new appeal by filing a notice of appeal under 37 CFR 41.31 followed by an appeal brief under 37 CFR 41.37. The previously paid notice of appeal fee and appeal brief fee can be applied to the new appeal. If, however, the appeal fees set forth in 37 CFR 41.20 have been increased since they were previously paid, then appellant must pay the difference between the increased fees and the amount previously paid.

A Supervisory Patent Examiner (SPE) has approved of reopening prosecution by signing below.

Response to applicant's arguments/remarks

Nonstatutory obviousness type double patenting rejection

This rejection is withdrawn in view of applicant's persuasive arguments.

REJECTION

Claim Rejections – 35 USC 112 – First Paragraph

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1, 5, and 7-8 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. Although the specification provides enablement for the instant claimed invention is enabled for methods of reducing elevated levels of PDE IV, or treating, a mammal suffering from **inflammatory respiratory conditions, specifically asthma and COPD,** comprising administering an effective amount of a compound of formula (I) to said mammal, it does not provide enablement for other inflammatory or autoimmune diseases. This is a scope of enablement rejection.

To be enabling, the specification of the patent application must teach those skilled in the art how to make and use the full scope of the claimed invention without undue experimentation. *In re Wright*, 999 F.2d 1557, 1561 (Fd. Cir. 1993). Explaining what is meant by “undue experimentation,” the Federal Circuit has stated that:

The test is not merely quantitative, since a considerable amount of experimentation is permissible, if its is merely routine, or if the specification in question provides a reasonable amount of guidance with respect to the direction in which experimentation should proceed to enable the determination of how to practice a desired embodiment of the claimed invention. PPG v Guardian, 75 F.3d 1558, 1564 (Fed. Cir. 1996).

The factors that may be considered in determining whether a disclosure would require undue experimentation are set forth in *In re Wands,* 8 USPQ2d 1400 (CAFC 1988) at 1404 wherein, citing *Ex parte Forman* 230 USPQ 546 (Bd Apls 1986) at 547 the court cited eight factors:

1) the quantity of experimentation necessary,
2) the amount of direction or guidance provided,
3) the presence or absence of working examples,
4) the nature of the invention,
5) the state of the prior art,
6) the relative skill of those in the art,
7) the predictability of the art, and
8) the breadth of the claims

These factors are always applied against the background understanding that scope of enablement varies inversely with the degree of unpredictability involved. *In re Fisher*, 57 CCPA 1099, 1108, 427 F.2d 833, 839, 166 USPQ 18, 24 (1970). Keeping that in mind, the Wands factors are relevant to the instant fact situation for the following reasons:

The nature of the invention

The invention in general is directed to a method of reducing elevated levels of PDE IV in a mammal, or a method of treating an inflammatory disease or an immune disease in a mammal, comprising administering an effective amount of a compound of formula (I).

The relative skill of those in the art

The relative skill of those in art is high, generally that of an M.D. or Ph.D.. However, to the extent that the instant disclosure provides only general guidance regarding the treatment of specific conditions associated with elevated PDE IV levels in humans, it is doubtful that an artisan skilled in the art would be able to practice the instant claimed invention commensurate with the scope of the claims in view of the state of the prior art as discussed below.

The state and predictability of the art

Sommer et al. (Sommer et al. Therapeutic potential of phosphodiesterase type 4 inhibition in chronic autoimmune demyelinating disease. Journal of Neuroimmunology. **1997**;79:54-61; already made of record by applicant) teach that selective PDE-4 suppresses the clinical manifestations of acute experimental autoimmune encephalomyelitis (EAE) in an animal model of multiple sclerosis (MS) and inhibits the production of tumor necrosis factor-alpha (abstract).

Bielekova et al. (Bielekova et al. Therapeutic potential of phosphodiesterase-4 and 3- inhibitors in Th1-mediated autoimmune diseases. The Journal of Immunology.

2000; 164: 1117-1124; already made of record) teach that hyperactive Th1-mediated responses are thought to be involved in the pathogenesis of many autoimmune diseases, including multiple sclerosis (page 1117). Bielekova et al. state that PDE4, as well as PDE3. inhibitors exert complex immunomodulatory properties, including inhibiting Ag-mediated T cell proliferation as well as skew the T cell cytokine profile toward a Th2 phenotype by down-regulating the expression or production of Th1 cytokines (page 1117, col. 1, first para.). Bielekova et al. state that PDE4 inhibitors have no effect on Th2 cytokines in animals (page 11117, col. 1, lines 10-15). Bielekova et al. disclose in vitro data showing that PDE4 inhibition Bielekova et al. state that even though PDE inhibitory properties of PDE4 inhibitors render said compounds as candidates for Th1-mediated autoimmune disorders (e.g. experimental encephalomyelitis models and collagen-induced arthritis models), the simple extrapolation of therapeutic efficacy from animal models to human disorders is not easily feasible (page 1117, col. 1, last ten lines).

Burnouf et al. (Burnouf et al. Recent advances in PDE4 inhibitors as immunoregulators and anti-incompetent cell activation by selective PDE4 inhibitors, supporting their potential use as emerging therapies in various inflammatory and immune diseases (page 1256, col. 2, last para.). Burnouf et al. state that a few clinical trials have confirmed the hypothesis that PDE4 inhibitors may provide benefit in the treatment of chronic airway diseases, such as asthma and COPD (page 1256, last para.). Burnouf et al. disclose that PDE4 activity is associated with a wide variety of diseases some of which have been related to an inflammatory state (e.g. asthma, chronic obstructive pulmonary diseases (COPD), rheumatoid arthritis (RA), while others have recently been connected to autoimmune pathology (abstract). Burnouf et al. disclose that **the clinical use of PDE4 prototype inhibitor drugs have been limited due to the side effects** that occur at therapeutic plasma levels (abstract; and page 1258, col. 1, last four lines). Based on the teaching of Burnouf et al. that the clinical use of PDE4 inhibitors has been limited due to side effects that occur at therapeutic levels, it is doubtful that results of in vitro methods disclosed by applicant to determine the PDE, TNF, and NFkB activity of the instant claimed compounds could be extrapolated to the clinical setting to reliably and predictably treat a mammal with other inflammatory conditions (such as multiple sclerosis, or Chron's, viral infections) or other autoimmune conditions (such as systemic lupus erythematosus (SLE)) because in vitro PDE inhibition studies have not been shown to correlate with the type of responses observed in humans treated with PDEIV inhibitors as evidenced by the teaching of Burnouf et al. that human subjects treated with PDE4 inhibitors experience side effects at therapeutic plasma levels of PDE IV inhibitors (abstract; and page 1258, col. 1, last four lines).

Further, Dastidar et al. (Dastidar et al. Therapeutic benefit of PDE4 inhibitors in inflammatory diseases. Current Opinion in Investigational Drugs. **2007**;8(5):364-372. Dastidar et al) teach that phosphodiesterase enzymes (PDEs) are cyclic nucleotide hydrolyzing enzymes that are spatially distributed in various tissues (page 364, col. 1, last para., lines 1-2). Dasitdar et al. state that there is a wide distribution of these enzymes inflammatory cells critical to the pathogenesis of inflammatory diseases, including asthma and COPD, that preferentially express PDE4 (page 364, col. 1, last para., lines 2-5). Dastidar et al. also teach that inflammation is the basic response to infection and/or external and internal injury and that both inflammatory and autoimmune diseases are often associated with deregulated expression and biosynthesis of inflammatory cytokines, which influence a plethora of cellular functions (page 364, col. 2, last full para.). Dastidar et al. state that down-modulation of an inflammatory signal concomitant with amplification of a counter anti-inflammatory signal would provide a rational approach to the treatment of inflammatory diseases (page 364, col. 2, lass full para.). Also, Dastidar et al. disclose that **contradictory evidence exist on the effects of PDE4 inhibition on cytokine production** and that whether PDE4 inhibitors have increased therapeutic potential in typical Th1 (e.g. rheumatoid arthritis and inflammatory bowel disease) or Th2 (e.g. asthma and atopic dermatitis) mediated diseases is still unclear (page 365, col. 2, first full para.). Dastidar et al. state that it must be noted that **animal models of arthritis are induced and do not occur naturally and therefore are not truly reflective of human pathogenesis** (page 366, col. 2, first full para.). Dastidar et al. state that while preclinical data often support the clinical application of PDE4 inhibitors, **the translation of this preclinical efficacy to success in trials can prove difficult** (page 366, col. 2, first full para.). Dastidar et al. state that a variety of cytokines such as TNF-alpha, IL-1-beta, IL-6, and IL8 contribute to immune stimulation and cause intestinal damage and T-helper cell differentiation (page 366, col. 2, last para.). Also, Dastidar et al. state that chronic inflammatory bowel disease (IBD e.g. Chron's disease) utilize nonspecific anti-inflammatory and immunosuppressive agents that are only partially effective and have serious side effects (page 366, col. 2, last para.). Dastidar et al. state that PDE4 inhibitors have been investigated for the treatment of age-related memory impairment, Alzheimer's disease, as well as skeletal muscle atropy, hypertension, and autoimmune diseases; however, **their therapeutic benefits in these indications have yet to be demonstrated in the clinical setting** (page 367, col. 2, second full para.). Further, Dastidar et al. state that the clinical efficacy of PDE4 inhibition is more concrete for Th2-mediated respiratory disorders and that whether this will also benefit patients with Th1-mediated disorders remains to be established (page 369, last para). Dastidar et al. disclose that even though preclinical evidence suggest that this might well be the case, PDE4 inhibition still requires further clinical investigation, especially when considering the possible side effect profiles associated with systemic exposure (page 369, last para. to page 370, first three line s). Since Dastidar suggest that there is a plethora of de-regulated, or unregulated, PDE-IV related human conditions that could be potentially treated with a PDE-IV inhibitor (e.g. **asthma, COPD, arthritis, inflammation in response to infection or internal/external injury**; page 364, col. 1, last para.), it is doubtful that an artisan skilled in the would be able to practice the instant claimed invention commensurate in scope with the claims in view of the teaching of Dastidar et al. that animal models of arthritis are not truly reflective of human pathogenesis (page 366, col. 2, first full para.) and that while preclinical data often support the clinical application of PDE4 inhibitors, the translation of this efficacy to success in clinical trials can prove difficult (page 366, col. 2, first full para.). Besides, Dastidar et al. disclose that the therapeutic benefits of PDE4 inhibitors in treatment of age-related memory impairment, Alzheimer's disease, as well as skeletal muscle atropy, hypertension, and **autoimmune diseases** are yet to be demonstrated in the clinical setting (page 367, col. 2, second full para.).

It is noted that cilomilast (Ariflo) is a PDE-IV inhibitor that was approved by the US FDA at the end of 2002 for use in maintenance of lung function in COPD patients (Anonymous. Ariflo (Cilomilast)- Oral PDE-IV Inhibitor for Chronic Obstructive Pulmonary Disease (COPD). www.drugdevelopment-technology.com/projects/**ariflo.** Electronic pages 1-2). It is also disclosed that several investigational PDE-IV drugs have caused excessive nausea and their development discontinued (page 2, marketing commentary section).

Based on the state of the art at the time the instant application was filed, their is serious doubt that the combination of the instant disclosure and in vitro data showing that the compounds of formula I are effective in inhibiting PDE-IV would have provided sufficient guidance or direction to one of skill in the art to practice the instant invention commensurate with the scope of the claims as evidenced by the fact that the clinical development of other PDE-IV inhibitors had to be discontinued because of excessive adverse effects. Further, although the PDE-IV inhibitor, cilomilast (Ariflo), is approved for clinical use, its approval is limited to the treatment of COPD. Hence, one would not expect to reliably and predictably practice the instant invention in non-respiratory inflammatory conditions or autoimmune diseases without conducting extensive research in view of the state of the art in view of the fact that the prior art suggest the need for further clinical investigation of PDE-IV inhibitors in the treatment of non-respiratory inflammatory diseases and autoimmune diseases (e.g. Bielekova et al. state that even though PDE inhibitory properties of PDE4 inhibitors render said compounds as candidates for Th1-mediated autoimmune disorders (e.g. experimental encephalomyelitis models and collagen-induced arthritis models), the simple extrapolation of therapeutic efficacy from animal models to human disorders is not easily feasible, page 1117, col. 1, last ten lines; Burnouf et al. disclose that **the clinical use of PDE4 prototype inhibitor drugs have been limited due to the side effects** that occur at therapeutic plasma levels, abstract; and page 1258, col. 1, last four lines; and Dastidar disclose that the therapeutic benefits of PDE4 inhibitors are yet to be demonstrated in autoimmune diseases, page 367, col. 2, second full para.).

Scope of the Claims

Claim 1 recites "[a] method of reducing elevated levels of PDE IV in a mammal," which encompasses a plethora of PDE-4 associated condition since PDEIV is associated with inflammatory (e.g. viral infection; asthma, COPD) and autoimmune conditions (e.g. multiple sclerosis). However, Dastidar et al. disclose that the therapeutic benefits of PDE4 inhibitors in treatment of age-related memory impairment, Alzheimer's disease, as well as skeletal muscle atropy, hypertension, and **autoimmune diseases** are yet to be demonstrated in the clinical setting (page 367, col. 2, second full para.). Further, claims 1 and 5 recite the term "an effective amount of a compound of formula (I)." However, a dose that is effective to reduce PDE IV levels in a mammal may not necessarily be an amount that is effective to treat an inflammatory disease or an autoimmune disease in a mammal.

The amount of direction or guidance provided and the presence or absence of working examples

The specification provides limited direction or guidance for determining the particular administration regimens (dosages, timing, administration routes) necessary to treat all inflammatory and autoimmune diseases associated with unregulated angiogenesis. Further, the working examples are directed on making the claimed compounds an in vitro experiments. No specific in vivo data is disclosed to establish any correlation between the in vitro examples and the contemplated therapeutic effects to be achieved in practicing the claimed methods in a human and the clinical use of PDE-IV inhibitors has been limited to treating respiratory conditions. Thus, applicant at best has provided specific direction or guidance only for an in vitro methodology for evaluating PDE IV inhibitory effects of the claimed compounds of formula I without any reasonable specific guidance for use of the compounds of formula I for treating non-respiratory inflammatory conditions or autoimmune diseases.

The quantity of experimentation necessary

In view of applicant's disclosure, coupled with the plethora of inflammatory and autoimmune diseases that are associated with elevated PDE IV levels (specification, page 24, lines 23-26) and as evidenced by the teaching of the prior art (see Distadar et al., page 364, col. 1, last para.), it is reasonable to surmise that the level of uncertainty and unpredictability in the art regarding extrapolation of preclinical data to the clinical setting would reasonably require one skilled in the art to conduct more than routine experimentation in order to practice the claimed invention commensurate in scope with the claims.

Response to applicant's arguments

In response to applicant's arguments set forth in the Appeal Brief that the claims are enabled because the specification contains a teaching of the manner and process of making and using an invention in terms which correspond in scope to those used in describing and defining the subject matter sought to be patented and that all that is all that is required for those of ordinary skill in the art to practice the claimed invention by conducting routine experimentation, it is the examiner's position that more than routine experimentation would be required to practice the instant claimed invention commensurate with the scope of the claims in view of the specification as originally filed because of the unpredictability and unreliably of the state of the art as evidenced by the following:

a) Bielekova et al. state that even though PDE inhibitory properties of PDE4 inhibitors render said compounds as candidates for Th1-mediated autoimmune disorders (e.g. experimental encephalomyelitis models and collagen-induced arthritis models), the simple extrapolation of therapeutic efficacy from animal models to human disorders is not easily feasible, page 1117, col. 1, last ten lines;

b) Burnouf et al. disclose that **the clinical use of PDE4 prototype inhibitor drugs have been limited due to the side effects** that occur at therapeutic plasma levels, abstract; and page 1258, col. 1, last four lines; and

c) Dastidar disclose that the therapeutic benefits of PDE4 inhibitors are yet to be demonstrated in autoimmune diseases, page 367, col. 2, second full para.).

d) The clinical use of the PDE-IV inhibitor, cilomilast, is limited for use in the treatment of COPD and other PDE-IV inhibitors that were potential candidates for clinical use were discontinued from development because of their toxicity.

e) Although Sommer et al. (Sommer et al. Therapeutic potential of phosphodiesterase type 4 inhibition in chronic autoimmune demyelinating disease. Journal of Neuroimmunology. 1997;79:54-61; already made of record by applicant) teach that selective PDE-4 suppresses the clinical manifestations of acute experimental autoimmune encephalomyelitis (EAE) in an animal model of multiple sclerosis (MS) and inhibits the production of tumor necrosis factor-alpha (abstract), this reference predates the above references. Hence, it is the examiner's position that Sommer et al. is not truly reflective of the state of the art in view of Bielekova et al. and Dastidar et al.

Regarding applicant's argument that it is erroneous to rely on Bielekova et al. that "simple extrapolation of therapeutic efficacy from animal models to human disorders is not easily feasible," it is the examiner's position that said statement is consistent with the state of knowledge in the art regarding PDE-IV inhibitors as evidenced by the above discussion regarding the state of the art. Besides, Bielekova et al. state that well designed therapeutic trials supported by immunological studies should not only provide more definite information about the therapeutic potential of PDE inhibitors in autoimmune disorders, but also broaden our understanding of the immunopathogenesis and the potential differences among individual human THl-mediated autoimmune diseases (page 1123, column 2, last paragraph), which is considered an invitation for extensive clinical research. Although applicant is not required to prove the ultimate value in humans of the asserted utility of compounds of formula (I), the fact that the development of other PDE-IV inhibitors has been discontinued from development coupled with the fact the clinical indication of the first PDE-IV inhibitor, approved in the US (in 2002) was limited to treatment of COPD creates serious doubt that the instant claimed compounds of formula (I) by virtue of their PDE-IV inhibitory activity would predictably and reliably be use to treat non-respiratory inflammatory conditions and autoimmune diseases in view of the state of the art at the time the application was originally filed. Hence, it is the examiner's position that applicant has not provided any information in the specification that specifically establish a correlation between the specific chemical compounds of formula I and their clinical effects in treating or reducing elevated PDE-IV levels in the non-respiratory inflammatory and autoimmune diseases encompassed by the claims when viewed in light of the specification.

Also, Applicant's argument that the references of Burnouf et al., Dastidar et al., and Sommer et al. demonstrate the therapeutic potential of PDE IV inhibition in a wide variety of autoimmune or inflammatory diseases, and thus, the predictive value of the test results provided by applicant is sufficiently established by these references is not found to be persuasive because it is the examiner's position that these references provide an invitation for further clinical investigation of PDE-IV inhibitors for use in the treatment of non-respirator inflammatory conditions and autoimmune diseases for the reasons discussed above and not actual clinical ultility.

In response to applicant's statement that is unclear what limitations of claim 1 are referred to by the Examiner as encompassing "various diverse condition since claim 1 recites "reduction of PDE IV," it is noted that the term "reduction of PDE IV" when viewed in light of the specification clearly encompasses a plethora of inflammatory and autoimmune disorders (specification, pages 23-24). However, as discussed above, there is serious doubt that PDE-IV inhibitors by virtue of the inhibitory effects alone can be reliably and predictably be used to treat non-respiratory inflammatory conditions and autoimmune diseases.

Further, in view of the clinical unreliably and unpredictability of PDE-IV inhibitors, it is the examiner's position that "how the chemical feature of a compound relates to the immunpathologies of the disease the compounds purports to treat" is not completely irrelevant or contrary to In re Brana for patentability purposes because there is serious doubts that a showing of PDE-IV inhibitory in vitro activity alone is sufficient to reasonably and predictably determine the clinical usefulness of PDE-IV inhibitor drugs in view of the prior art as discussed above.

Thus, it is the examiner's position that an artisan skilled in the art would have to conduct undue experimentation in order to practice the instant claimed invention commensurate with the scope of the claims.

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Charlesworth Rae whose telephone number is 571-272-6029. The examiner can normally be reached between 9 a.m. to 5:30 p.m. Monday to Friday.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Sharmila G. Landau, can be reached at 571-272-0614. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR.

Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http:pair-direct.uspto.gov. Should you have any questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 800-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

5 May 2009
/C. R./ Examiner, Art Unit 1611

/Sharmila Gollamudi Landau/
Supervisory Patent Examiner, Art Unit 1611